Complete Summary

GUIDELINE TITLE

Estrogen and progestogen use in peri- and postmenopausal women: September 2003 position statement of The North American Menopause Society.

BIBLIOGRAPHIC SOURCE(S)

Estrogen and progestogen use in peri- and postmenopausal women: September 2003 position statement of The North American Menopause Society. Menopause 2003 Nov-Dec;10(6):497-506. [71 references] PubMed

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

- Perimenopause
- Postmenopause

GUIDELINE CATEGORY

Management
Treatment

CLINICAL SPECIALTY

Cardiology
Endocrinology
Family Practice
Geriatrics
Internal Medicine
Obstetrics and Gynecology
Oncology

INTENDED USERS

Health Care Providers
Physician Assistants
Physicians

GUIDELINE OBJECTIVE(S)

To present clinical recommendations for use of hormone therapy (HT) in peri- and postmenopausal women

TARGET POPULATION

Peri- and postmenopausal women

INTERVENTIONS AND PRACTICES CONSIDERED

Menopause-related hormone therapy, including:

1. Estrogen therapy (ET)
2. Systemic ET/estrogen-progestogen therapy (EPT)
3. Local ET
4. Progestogen therapy (progesterone and progestin)

MAJOR OUTCOMES CONSIDERED

The risk-benefit ratio of postmenopausal estrogen therapy (ET) and estrogen-progestogen therapy (EPT) for both disease prevention and treatment of specific menopause-related symptoms

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

The level of evidence indicated for each study is based on a grading system that evaluates the scientific rigor of the study design, as developed by the U.S. Preventive Services Task Force.

Levels of Evidence

Level I Properly randomized, controlled trial

Level II-1 Well-designed controlled trial but without randomization

Level II-2 Well-designed cohort or case-control analytic study, preferably from more than one center or research group

Level II-3 Multiple time series with or without the intervention (e.g., cross-sectional and uncontrolled investigational studies); uncontrolled experiments with dramatic results could also be regarded as this type of evidence

Level III Opinions of respected authorities that are based on clinical experience; descriptive studies and case reports; reports from expert committees

METHODS USED TO ANALYZE THE EVIDENCE

Review
Review of Published Meta-Analyses

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

The North American Menopause Society (NAMS) Board of Trustees convened a second Hormone Therapy Advisory Panel to develop an updated report on hormone therapy.

The 2003 Panel utilized the 2002 Hormone Therapy Advisory Panel report as a starting point. A two-part set of clinical questions was developed by the Panel. The first set related to items for which complete agreement was previously reached; the second set related to areas of previous nonconsensus. Each Panelist completed the questionnaire on a blinded basis (i.e., unaware of the responses of the other Panelists). The responses were collated in the North American Menopause Society Central Office, again into two lists: those with consensus and those without. All responses were distributed to the entire Panel.

The Panel reviewed all of the responses by telephone conference call in an attempt to reach consensus. Further development of the report through multiple drafts was conducted through the Internet. The clinical recommendations indicate where consensus was achieved as well as where opinions differed.

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

The position statement was reviewed and approved by The North American Menopause Society (NAMS) 2002-2003 Board of Trustees.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Recommendations for Clinical Practice: Areas of Consensus

The Panel agreed on the following clinical recommendations for postmenopausal hormone therapy:

- A strong recommendation was made for uniform and consistent terminology for menopause-related therapies, as indicated below:

  ET: Estrogen therapy

  EPT: Combined estrogen-progestogen therapy

  HT: Hormone therapy (encompassing both ET and EPT)

  CC-EPT: Continuous-combined estrogen-progestogen therapy (daily administration of both estrogen and progestogen)

  CS-EPT: Continuous-sequential estrogen-progestogen therapy (estrogen daily, with progestogen added on a set sequence)

  Systemic ET/EPT: Preparations of ET or EPT that have a systemic, not solely vaginal, effect

Local ET: Preparations of ET that have a predominately vaginal, not systemic, effect

Progestogen: Encompassing both progesterone and progestin

- Treatment of moderate to severe menopause symptoms (i.e., vasomotor symptoms, sleep disruption from vasomotor symptoms) remains the primary indication for systemic ET and EPT. Every systemic ET/EPT product is government approved for this indication.
- Every systemic and local ET/EPT product is government approved for treating moderate to severe symptoms of vulvar and vaginal atrophy, such as vaginal dryness, dyspareunia, and atrophic vaginitis. When hormones are considered solely for this indication, local ET is generally recommended.
- The primary menopause-related indication for progestogen use is endometrial protection from unopposed ET. For all women with an intact uterus who are using estrogen therapy, clinicians are advised to prescribe adequate progestogen, in either a CC-EPT or CS-EPT regimen. Women without a uterus should not be prescribed a progestogen.
- Some women with an intact uterus who choose EPT may experience undesirable side effects from the progestogen component. However, there is insufficient evidence regarding long-term endometrial safety to recommend use of long-cycle progestogen (i.e., progestogen every 3–6 months for 12–14 days), a progestin-containing intrauterine device (IUD), or low-dose estrogen without progestogen as an alternative to standard EPT regimens. If utilizing any of these approaches, closer surveillance of the endometrium is recommended, pending more definitive research.
- No EPT regimen should be used for primary or secondary prevention of coronary heart disease (CHD) or stroke.
- The effect of ET on coronary heart disease and stroke is not yet clear. ET does not have a significant effect on stroke risk in postmenopausal women with known ischemic cerebrovascular disease, but for healthy older women, effects of ET on stroke risk are not clear. However, unless confirming data become available, ET should not be used for primary or secondary prevention of these conditions.
- Breast cancer risk is increased with ET and, to a greater extent, EPT use beyond 5 years. Progestogen appears to contribute substantially to that adverse effect. EPT and, to a lesser extent, ET increase breast cell proliferation, breast pain, and mammographic density. HT may impede the diagnostic interpretation of mammograms. One recent observational study suggests that the increase in incidence of breast cancer with oral, transdermal, and implanted estrogens varies little between specific estrogens and progestogens or their doses, or between continuous and sequential regimens. The observational data also suggest that breast cancer incidence may begin to increase slightly with less than 5 years HT use. Observational data from one study suggest that HT use may be associated with increased breast cancer mortality, but insufficient data exist to determine whether ET or EPT, or duration of use of ET or EPT, is associated with any increase in mortality.
- There is definitive evidence for EPT efficacy in reducing risk for postmenopausal osteoporosis fracture. There is, to date, no comparable evidence for ET. Many EPT and ET products are government approved for prevention of postmenopausal osteoporosis (i.e., loss of bone mineral

density) through long-term treatment. Because of the potential risks associated with HT, for women who require drug therapy for osteoporosis risk reduction (including women at high risk of fracture in the next 5–10 years), alternatives to HT should also be considered, weighing the risks and benefits of each. Recognition should be given to the fact that there are no published data on osteoporosis drug use beyond 7 years.

- Initiating EPT after age 65 cannot be recommended for primary prevention of dementia, as it increases the risk of dementia during the ensuing 5 years in this population. The evidence is insufficient to either support or refute the efficacy or harm of ET/EPT for primary prevention of dementia when therapy is initiated during the menopause transition or early postmenopause. However, given other adverse events that may be expected to accrue during long-term HT use, it is by no means clear that theoretical dementia benefits would outweigh known risks. HT does not appear to convey direct benefit or harm for secondary prevention (i.e., symptomatic treatment) of dementia due to Alzheimer's disease.
- The effects of HT on risk for breast cancer and osteoporotic fracture in symptomatic perimenopausal women have not been established in randomized clinical trials. The findings from trials in different populations (e.g., Women's Health Initiative [WHI]) should, therefore, be extrapolated with caution. There is, however, no evidence that symptomatic women differ from asymptomatic women in cancer or bone outcomes.
- Data from studies such as the WHI and the Heart and Estrogen/progestin Replacement Study (HERS) should be extrapolated only with caution to women younger than 50 years of age who initiate hormone therapy. WHI and HERS involved women aged 50 and over (with mean ages of 63 and 67, respectively), and HERS was conducted solely in women with known coronary artery disease. The data should not be extrapolated to women experiencing premature menopause (<40 years of age) and initiating hormone therapy at that time.
- Premature menopause and premature ovarian failure are conditions associated with earlier onset of osteoporosis and coronary heart disease, but there are no clear data as to whether ET or EPT will reduce morbidity or mortality from these conditions. The benefit-risk ratio may be more favorable for younger women.
- Use of ET and EPT should be limited to the shortest duration consistent with treatment goals, benefits, and risks for the individual woman, taking into account symptoms and domains (e.g., sexuality, sleep) that may have an impact on quality of life.
- Lower-than-standard doses of ET and EPT should be considered (i.e., daily doses of 0.3 mg conjugated estrogens tablet, 0.25 to 0.5 mg micronized 17beta-estradiol tablet, 0.025 mg 17beta-estradiol patch, or the equivalent). Many studies have demonstrated nearly equivalent vasomotor and vulvovaginal symptom relief and preservation of bone mineral density. Lower EPT doses are better tolerated and may or may not have a more positive safety profile than standard doses; however, lower doses have not been tested for outcomes (including endometrial safety) in long-term trials.
- Nonoral routes of administration of ET/EPT may offer advantages and disadvantages, but the long-term benefit-risk ratio has not been demonstrated. Differences would be related to the role of the first-pass hepatic effect, the hormone concentrations in the blood achieved by a given route, and the biologic activity of component ingredients. There is some evidence that transdermal 17beta-estradiol does not increase the level of C-

reactive protein, and also that it may be associated with lower risk of deep venous thrombosis than oral estrogen. A large observational study has shown similar increased risks for breast cancer with both oral and transdermal estrogens.

- Extended use of ET or EPT is acceptable under the following circumstances, provided the woman is well aware of risks and there is strict clinical supervision:
    - For the woman for whom, in her opinion, benefits of symptom relief outweigh risks, notably after failing an attempt to withdraw HT. Attempts should be made over time to reduce and cease HT.
    - For women with moderate to severe menopause symptoms who are at high risk for osteoporotic fracture. Attempts should be made over time to lower the dose or cease HT and introduce alternate bone-sparing therapy.
    - For prevention of osteoporosis in a high-risk woman when alternate therapies are not appropriate for that woman.
- Prior to consideration of any therapeutic regimen, including HT, all women should have a complete health evaluation, including a comprehensive history and physical examination. More specific examinations, such as bone densitometry, should be considered on a case-by-case basis.
- The Panel acknowledged that the absolute risks published thus far regarding ET/EPT are small (e.g., the EPT arm of the WHI), as are the benefits for bone and reduction in colon cancer risk. For women younger than 50 or those at low risk for coronary heart disease, stroke, osteoporosis, breast cancer, or colon cancer, the absolute risk or benefit from EPT is likely to be smaller than demonstrated in WHI, although the relative risk may be similar. An individual risk profile is essential for every woman contemplating any regimen of EPT or ET. Women should be informed of known risks.

Areas Where Insufficient or Conflicting Evidence Precludes Consensus

The Panel could not reach consensus on the following issues, but the summary of responses is of relevance to clinicians:

What are the currently acceptable definitions of "short-term" and "long-term" HT? The Panel could not reach a consensus regarding definitions of these terms, agreeing that delineating specific time periods is arbitrary and that no uniform time can be broadly applied to all women. The Panel recognized that this question is an attempt to assign a "safe window" for HT. The dilemma is that current data suggest that the risk of breast cancer is significantly increased beyond 5 years use, with a lower elevation in risk before 5 years, whereas there is evidence of potential early coronary heart disease and thromboembolism risk within the first 2 years of use and conflicting evidence of early risk of ischemic stroke. Moreover, there are emerging data showing no association of early increase in coronary heart disease events in young (i.e., average age 53), healthy postmenopausal women with HT during the first 2 years of treatment. However, deep venous thrombosis is slightly increased from an expected annualized rate of 0.3 per 1,000 to 0.9 per 1,000. It is therefore difficult to define any "safe window," and an individual risk-benefit profile needs to be considered for every woman considering commencement of hormone therapy.

Is HT associated with early risk of coronary heart disease?
Panelists were divided on the issue as to whether there is definitive evidence for early increased risk of coronary heart disease with HT. For women similar to participants in the combined EPT arm of the WHI (average age 63 years; range from 50 to 79 years), the WHI data are the best estimate of early harm from combined estrogen-progestogen therapy. The WHI demonstrated that EPT may increase the risk of coronary heart disease among generally healthy postmenopausal women during the first year after initiation of hormone use. There is also evidence that early harm within 2 years of use may not pertain to healthy menopausal women using ET/EPT for menopause symptom management.

How long should HT be prescribed for symptom relief?
No consensus could be reached, although a general guiding principle should be for the shortest time at the lowest possible dose. The Panel recognized that symptoms can recur when therapy is discontinued, independent of age and duration of HT use. Useful information regarding the consideration of reinstituting HT is anticipated from the terminated EPT arm of the WHI, as trial participants are being followed for outcomes after termination. The Panel agreed that the decision to reinstitute HT should be individualized based on severity of symptoms, current risk/benefit considerations, and the woman's preference. Reinstituting therapy at a lower dose may facilitate future attempts at discontinuing.

Is there a best way to discontinue HT?
Panelists were divided in their recommendations, including both abrupt therapy cessation and tapering the dose. Past history of severe symptoms may favor tapering, but no specific protocols could be recommended. Some gradually decrease the dose, while others lengthen the time between doses. Matrix transdermal HT patches can be trimmed to provide smaller doses. Current data are inadequate to suggest that one method is better than the other.

Is it possible to make general conclusions about all members of the estrogen and progestogen families?
The majority opinion was that it is not possible to extrapolate conclusions from the study of one compound directly to another. It was acknowledged that estrogen and progesterone agonists share some common features and effects, and the only way to establish definitively the net clinical outcome for any given agent (alone or in combination) is through randomized clinical trials. In the absence of clinical trial data for each estrogen and progestogen, the clinical trial results for one agent probably should be generalized to all agents within the same family, especially with regard to adverse effects.

Does a continuous-combined EPT regimen (CC-EPT) have an effect different from continuous estrogen with sequential progestogen (CS-EPT)?
There are some indications that continuous progestogen in the dosages administered in studies such as the WHI and HERS may be related to these trials' adverse cardiovascular and breast outcomes, but conflicting data preclude a consensus.

Does HT enhance quality of life (QOL)?
There is a lack of consensus on the impact of hormone therapy on quality of life. This has largely been due to a lack of agreement in the scientific community regarding how best to obtain an appropriate evaluation of quality of life, including

the domains to be incorporated into any survey instruments. There is consensus that validated instruments for determining the impact of hormone therapy, or indeed any menopause-related therapy, on quality of life should be incorporated into future studies.

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The position statement was supported by evidence from randomized, controlled clinical trials, meta-analyses, and review articles. If the evidence was contradictory or inadequate to form a conclusion, a consensus-based opinion was made.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Overall Benefits

- Appropriate use of hormone replacement therapy (HRT) in peri- and postmenopausal women to maximize health benefits while minimizing health risks
- Relief of moderate to severe menopause symptoms

Specific Benefits

There is definitive evidence for combined estrogen-progestogen therapy (EPT) efficacy in reducing risk for postmenopausal osteoporosis fracture.

Subgroups Most Likely to Benefit

- Women with moderate to severe menopause symptoms who are at high risk for osteoporotic fracture.
- High-risk women for whom alternate therapies for the prevention of osteoporosis are not appropriate.

POTENTIAL HARMS

Breast cancer risk is increased with estrogen therapy and, to a greater extent, combined estrogen-progestogen therapy use beyond 5 years. Progestogen appears to contribute substantially to that adverse effect. Combined estrogen-progestogen therapy and, to a lesser extent, estrogen therapy may increase breast cell proliferation, breast pain, and mammographic density. Hormone therapy may impede the diagnostic interpretation of mammograms.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

This position statement focuses on the use of government-approved prescription estrogen therapy/combined estrogen-progestogen therapy (ET/EPT) products available in the United States and Canada, not custom estrogen therapy/combined estrogen-progestogen therapy preparations, selective estrogen-receptor modulators (SERMs), or hormones available without a prescription (including phytoestrogens).

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Staying Healthy

IOM DOMAIN

Effectiveness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Estrogen and progestogen use in peri- and postmenopausal women: September 2003 position statement of The North American Menopause Society. Menopause 2003 Nov-Dec;10(6):497-506. [71 references] PubMed

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2002 Oct 6 (revised 2003 Sep)

GUIDELINE DEVELOPER(S)

The North American Menopause Society - Private Nonprofit Organization

SOURCE(S) OF FUNDING

The North American Menopause Society (NAMS)

GUIDELINE COMMITTEE

Hormone Therapy Advisory Panel

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

The Panel was composed of healthcare professionals from different areas of medical science related to the issue.

Advisory Panel Members

Wulf H. Utian, MD, PhD, FACOG (Chair) – Arthur H. Bill Professor Emeritus of Reproductive Biology and Obstetrics and Gynecology, Case Western Reserve University School of Medicine; Consultant in Gynecology, The Cleveland Clinic Foundation; President, Rapid Medical Research Inc., Cleveland, OH; NAMS Executive Director and Honorary Founding President, NAMS President 1989-1992, NAMS Board of Trustees 1989-Present

Peter Collins, MD, FRCP – Professor of Clinical Cardiology, Department of Cardiac Medicine, National Heart and Lung Institute, Faculty of Medicine, Imperial College London, London, UK

Bruce Ettinger, MD, FACP – Senior Investigator, Division of Research, Kaiser Permanente Medical Care Program, Oakland, CA; NAMS President 1996-1997, NAMS Board of Trustees 1993-1998

J. Chris Gallagher, MD – Professor of Medicine, Creighton University; Department of Metabolism, St. Joseph's Hospital, Omaha, NE; NAMS President 1994-1995, NAMS Board of Trustees 1990-1996 and 2002-Present

Margery L.S. Gass, MD – Professor of Clinical Obstetrics and Gynecology, University of Cincinnati College of Medicine; Director, University Hospital Menopause and Osteoporosis Center, Cincinnati, OH; NAMS 2002-2003 President, NAMS Board of Trustees 1999-Present; WHI and WHIMS Investigator

Morrie M. Gelfand, CM, MD – Professor of Obstetrics and Gynecology, McGill University; Honorary Chief, Department of Obstetrics and Gynecology, The Sir Mortimer B. Davis Jewish General Hospital; Co-Director, McGill University Menopause Clinic, Montreal, QC, Canada; NAMS 2001-2002 President, NAMS Board of Trustees 1997-2003

Victor W. Henderson, MD, MS – Professor of Geriatrics, Neurology, Pharmacology and Toxicology, and Epidemiology, Center on Aging, University of Arkansas for Medical Sciences, Little Rock, AR; NAMS Board of Trustees 2002-Present; Member, WHIMS External Advisory Board

David M. Herrington, MD, MHS – Professor of Internal Medicine/Cardiology, Associate in Public Health Sciences, Wake Forest University School of Medicine, Winston-Salem, NC; WHI and HERS Investigator

Marian C. Limacher, MD – Professor of Medicine, Division of Cardiovascular Medicine, University of Florida College of Medicine, Gainesville, FL; WHI and WHIMS Investigator

Rogerio A. Lobo, MD – Willard C. Rappleye Professor of Obstetrics and Gynecology, Columbia University College of Physicians and Surgeons, New York, NY; NAMS Board of Trustees 1989-1994

B. Lawrence Riggs, MD – Consultant in Endocrinology and Metabolism, Mayo Clinic and Foundation, Professor of Medicine, Mayo Medical School, Minneapolis, MN

Meir J. Stampfer, MD, DrPH – Professor of Epidemiology and Nutrition, Chair, Department of Epidemiology, Harvard School of Public Health, Boston, MA

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Panelist Disclosures

Chair, Wulf H. Utian, MD, PhD, FACOG – Industry consulting fees (Berlex, Eli Lilly, Endeavor, Pfizer, Roche, Warner Chilcott); direct industry lecture fees (none disclosed); industry research support (Amylin, 3M, Barr, Berlex, Bristol-Myers Squibb, Eli Lilly, Endeavor, Forest, Neurocrine Biosciences, Novartis, Novo Nordisk, Organon, Pfizer, Pharmacia, Procter & Gamble, Roche, Sepracor, Solvay, Wyeth, Yamanouchi)

Peter Collins, MD, FRP – Industry consulting fees (Eli Lilly, Novartis, Pfizer); direct industry lecture fees (Akzo Nobel, Merck, Novartis, Novo Nordisk, Schering, Wyeth); industry research support (Eli Lilly, Protein Technologies, Merck)

Bruce Ettinger, MD, FACP – Industry consulting fees (Berlex, Eli Lilly, Procter & Gamble, Tap); direct industry lecture fees (Berlex, Eli Lilly, Merck, Procter & Gamble, Tap); industry research support (Eli Lilly, Merck, Procter & Gamble)

J. Chris Gallagher, MD – Industry consulting fees (Aventis, Endeavor, Pfizer, Roche, Wyeth); direct industry lecture fees (Aventis, Organon, Pfizer, Roche, Wyeth); industry research support (Endeavor, Organon, Pfizer, Roche, Wyeth)

Margery L.S. Gass, MD – Industry consulting fees (Eli Lilly, GlaxoSmithKline, Merck, Procter & Gamble); direct industry lecture fees (Aventis); industry research support (Eli Lilly, GlaxoSmithKline, Merck, Pfizer, Procter & Gamble, Wyeth)

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Victor W. Henderson, MD, MS – Industry consulting fees (Wyeth); direct industry lecture fees (Wyeth); industry research support (none disclosed)

David M. Herrington, MD, MHS – Industry consulting fees (none disclosed); direct industry lecture fees (Eli Lilly, Organon, Pfizer, Wyeth); industry research support (Eli Lilly, Pfizer)

Marian C. Limacher, MD – Industry consulting fees (none disclosed); direct industry lecture fees (none disclosed); industry research support (Boehringer-Ingelheim, Wyeth)

Rogerio A. Lobo, MD – Industry consulting fees (Berlex, Merck, Novartis, Ortho-McNeil, Pfizer, Solvay, Wyeth); direct industry lecture fees (none disclosed); industry research support (Novartis, Wyeth)

B. Lawrence Riggs, MD – Industry consulting fees (Berlex); direct industry lecture fees (none disclosed); industry research support (Acologix)

Meir J. Stampfer, MD, DrPH – Industry consulting fees (Schering, Wyeth); direct industry lecture fees (none disclosed); industry research support (none disclosed)

Marcia L. Stefanick, PhD – Industry consulting fees (none disclosed); direct industry lecture fees (none disclosed); industry research support (none disclosed)

Nancy Fugate Woods, PhD, RN, FAAN – Industry consulting fees (Procter & Gamble); direct industry lecture fees (none disclosed); industry research support (none disclosed)

GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: North American Menopause Society. Amended report from the NAMS Advisory Panel on Postmenopausal Hormone Therapy. Menopause 2003 Jan-Feb;10(1):6-12.

GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from The North American Menopause Society (NAMS) Web site.

Print copies: Available from NAMS, P.O. Box 94527, Cleveland, OH 44101, USA. Order forms are available in Portable Document Format (PDF) from The North American Menopause Society Web site www.menopause.org.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This NGC summary was completed by ECRI on January 23, 2003. The information was verified by the guideline developer on February 13, 2003. This summary was updated by ECRI on March 5, 2004. The information was verified by the guideline developer on March 29, 2004.

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